HIGH-RISK PREGNANCY *and* DELIVERY

NURSING PERSPECTIVES

Elizabeth Stepp Gilbert, RN, MS

Judith Smith Harmon, RN, MS

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Elizabeth Stepp Gilbert, RN, MS
Assistant Professor,
Grand Canyon College,
Phoenix, Arizona

Judith Smith Harmon, RN, MS
Perinatal Clinical Nurse Specialist,
Good Samaritan Medical Center,
Phoenix, Arizona
Faculty, University of Phoenix, Phoenix, Arizona

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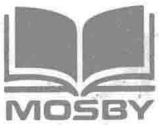

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Preface

The field of perinatal nursing is developing at a rapid pace. Out of this growth has come expanded knowledge leading to sophisticated care of the mother and fetus; the aim is to ensure an optimal outcome for the neonate and well-being of the mother while promoting the unity of the family. Not all high-risk pregnant women have ready access to a sophisticated perinatal obstetric service in a tertiary care center. Thus staff members in all obstetric care facilities, including primary care settings, must be prepared to screen for risk factors, render immediate stabilizing care in preparation for transport, and provide for preventive management. Nurses practicing in primary care settings must be able to assess and intervene rapidly and efficiently on the basis of scientific knowledge.

It is our conviction that thorough, sophisticated care of the maternal-fetal unit by all obstetric nurses can considerably decrease neonatal morbidity and mortality and lessen the incidence of later complications in children. Perinatal texts usually devote considerable attention to the care of the sick neonate; the intent of this text is to fill the void of nursing care knowledge of the high-risk maternal-fetal unit to improve the neonatal outcome.

This book is designed to provide obstetric nurses, practitioners, clinicians, and upper-division undergraduate and graduate students a text and reference for rendering high-risk maternal and fetal nursing care. The format was selected to provide for concise and systematic retrieval of information, focusing on knowledge beyond basic understanding. Additional references and resources are included for the reader who desires further information.

High-Risk Pregnancy and Delivery: Nursing Perspectives focuses on the physiologic and psychologic stressors of a high-risk pregnancy as they relate to the mother and the fetus. It is designed to provide the reader with a strong background in physiologic and pathophysiologic changes in pregnancy, their effects on the mother and fetus, and their potential effects on the neonate if optimal care is not implemented. Psychologic concepts are emphasized in one special unit and are interwoven throughout the text.

The text is organized into units and chapters based on physiologic adaptations to pregnancy, identification of high-risk pregnancy, antepartum and intrapartum assessment, psychologic implications of high-risk pregnancy, complicating conditions in pregnancy, and labor disorders. The content within the

chapters is organized according to the following format: incidence, etiology, and physiology, including pathophysiology and maternal and fetal/neonatal effects. The usual medical management is summarized so the nurse will be familiar with the possible plans of care and can thereby prepare and support the patient in considering her available options. A strong emphasis on the nursing process is a primary focus of each chapter. A major role of the nurse when caring for the expectant mother and her unborn child is one of prevention and early recognition; therefore patient education, assessment, intervention, and evaluation are heavily emphasized. Most chapters also include a suggested plan of nursing care, including potential patient problems, outcome criteria, and assessments and interventions. Other reference guides and tables are provided to consolidate critical information into a concise form.

ELIZABETH STEPP GILBERT
JUDITH SMITH HARMON

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UNIT I

PHYSIOLOGIC CONSIDERATIONS OF A HIGH-RISK PREGNANCY

Significant changes occur in female physiology following impregnation. Within a very few weeks alterations are measurable in many body systems. The mother must acquire and circulate increased quantities of nutrients to herself and the conceptus. Her body must form and maintain a new organ, the placenta. Through this, her body must dispose of fetal waste products and provide for fetal nutrition and respiration. Throughout the pregnancy, homeostatic mechanisms must adapt to provide for protection of the fetus and to guard against its rejection as a foreign substance.

When caring for the high-risk pregnant woman, the nurse must consider numerous physiologic aspects of pregnancy, such as adaptations in maternal body functions and the development of the maternal-fetal unit. Through analysis of these, specific antenatal assessments can be made and high-risk pregnancies can be identified. Special antepartum and intrapartum assessments of fetal well-being, including fetal monitoring and other surveillance modes, can also be instituted.

CHAPTER 1

Physiologic adaptations to pregnancy

In the sections to follow, adaptions in body functions will be described as they relate to cardiovascular, respiratory, metabolic, glandular, renal, hepatic, hematologic, gastrointestinal, and reproductive physiology. Descriptions of development of the fetus, beginning as an embryo, and the formation of the placenta will follow. Antepartum nursing assessments directed at promotion and maintenance of health will be described as they relate to laboratory studies, physical assessment, and nutritional requirements.

ADAPTATIONS IN BODY FUNCTIONS

Cardiovascular adaptations

Body fluid is compartmentalized into cellular space, interstitial space, and blood space. Water and solutes exit from the capillaries into the interstitial space where they are continually exchanged with water and solutes in the cellular space.

Blood, which consists of a suspension of various specialized cells in plasma, exerts a pressure on vessel walls. The pressure exerted depends on blood volume and distensibility of the vessel walls. Blood flow is related to the pressure gradient and the resistance of the vessel walls. Flow is measured as cardiac output and the pressure gradient is measured as arterial blood pressure. During pregnancy specific adaptations occur in these functions. These adaptations are ultimately aimed at fetal sustenance.

Total body fluid increases during pregnancy, but blood volume, in particular, increases by approximately 45% over prepregnant levels. The increase occurs in the cells and in the plasma of the blood. Plasma volume expands roughly 2.5 L to a total of 3.7 L. This increase in plasma volume results in increased intracapillary pressure and decreased oncotic pressure. The red blood cell mass may not increase in as great a proportion as the plasma.

To respond to the enlargement of the vascular volume, greater demands are placed on the heart to promote increased blood flow. Cardiac output rises from 4.5 to 5.5 L/min to 6 to 7 L/min. Cardiac output is a combination of stroke volume and heart rate; this is increased by 10 to 15 beats per minute (bpm).

Theoretically it might be assumed that arterial pressure would increase, but it does not because of hormonal and neurogenic influences. Both estrogen and the autonomic nervous system (ANS) promote vasodilation. Therefore arterial pressure actually decreases, with the diastolic pressure decreasing more than the systolic pressure. Venous pressure, however, increases as a reflection of resistance to blood return from the lower limbs because of partial obstruction of femoral veins.

Provided that diseases or pregnancy complications do not affect cardiac function, blood volume changes, or peripheral vascular resistance, the uteroplacental unit serves as a reservoir for the additional volume. Estimates of uterine blood flow are from 500 to 750 ml/min by term. Conditions such as cardiac disease, anemia, or hypertension can cause profound deficiencies in adaptive responses. Influences such as changes in the autonomic nervous system can also have profound effects on vasoconstrictive factors. Such changes may be seen with anesthesia. Simple changes in posture can reduce cardiac output. Obstruction of the inferior vena cava occurs when the pregnant woman is placed in a supine position.

In fact, at all times of crisis the uteroplacental unit is considered expendable to the mother. Any force competing against the cardiovascular adaptations will surely reduce blood flow to the uteroplacental unit and thus the fetus.

Respiratory changes

Respirations occur so that oxygen can be utilized in chemical processes for cellular proliferation and maintenance. This is accomplished through the processes of ventilation and gas transport and exchange between lungs, blood, and tissues.

Blood gases, primarily carbon dioxide and oxygen, remain in solution because of their individual or partial pressures. The partial pressures of each are changed in the alveoli of the lungs during inspiration and expiration. The gases move from the side of greater concentration to the side of lesser. This exchange occurs again between blood and tissues. Normal partial pressure of oxygen (pO_2) is 80 to 100 mm Hg and that of carbon dioxide (pCO_2) is 40 mm Hg. Oxygen, because of its low solubility, is carried by hemoglobin. Most carbon dioxide is carried in the plasma, becoming carbonic acid and increasing free hydrogen ions.

Since pregnancy increases cell numbers, requiring increased oxygen, changes are directed at facilitating oxygen availability. Hormonal influences and anatomic changes accomplish this.

Progesterone stimulates respiratory rate. The mother, however, increases her rate in excess of oxygen demand. In the process excess carbon dioxide is blown off. The resulting decrease in carbonic acid creates a pH difference in preg-

nancy. Normal pH is 7.38 to 7.42; in pregnancy the pH tends to rise to 7.40 to 7.42.

Anatomic changes also influence respiration. The upward displacement of the diaphragm by the gravid uterus causes a lateral expansion of the chest wall. Hormonal influences are thought to dilate the airways and to cause increased chest wall elasticity, which promotes these anatomic changes.

Changes in metabolism

Substrates for cellular metabolism are derived from ingested foods. They consist of carbohydrates, lipids, proteins, vitamins, and inorganic substances. These substrates are utilized to form new cells, to synthesize new substances, or to be burned as fuel for energy.

Carbohydrate is normally present in the blood primarily in the form of galactose and fructose. Galactose is converted to glucose, and fructose goes directly through the same pathways as glucose. Glucose can be utilized directly or can be converted and stored as glycogen. When glucose is not available, amino acids are the main source of glucose.

Glucose can be oxidized to carbon dioxide and water. Glucose can also be changed to glucose-6-phosphate, then channeled into glycogen formation or synthesized into other metabolites through degradation to pyruvate. Pyruvate enters the tricarboxylic Krebs cycle, in the presence of oxygen, and is converted to acetyl-CoA. Acetyl-CoA can also be formed by amino acids and fatty acids. Thus the Krebs cycle is a common junction in the metabolism of fats, proteins, and carbohydrates.

Lipids are hydrolyzed to form glycerol and fatty acids. Fatty acids are also synthesized from glucose and keto acids and can be stored as depot fat in the adipose tissue. When carbohydrates are not available, depot fat is mobilized so that fatty acids, easily oxidized, can be utilized as energy by cells. Fatty acids can also enter the Krebs cycle. In the liver, fatty acids may be used, in the absence of glucose, to spare amino acids. When this occurs, ketones are produced by fatty acid breakdown and are used as an alternative energy source.

Proteins are hydrolyzed to form amino acids. Amino acids are used for building new protein or for forming other nitrogen compounds. In order to supply energy, amino acids must be converted to carbohydrates or fats. Liver cells provide their own energy by converting amino acids into a carbohydrate form, keto acids, for entry into the Krebs cycle.

Insulin is necessary for utilization of glucose in oxidation and for the formation of glycogen and fats. It provides a carrier system for taking glucose into the cells and across the cellular membrane.

During pregnancy, there is an acceleration in the use of glucose because of rapid fetal cell and organ growth requiring a rapid source of energy. Compli-

cating this is a diminished maternal sensitivity to insulin. As a result pregnancy has been said to produce a diabetogenic state.

Because the fetus is a continuous feeder from the mother, who is a periodic feeder, a starvation-like situation increases the potential for ketonemia. Placental hormones contribute to this by promoting insulin resistance and forcing the woman into utilizing fats for energy needs. The entire metabolic rate increases in pregnancy, as does the need for increased caloric intake to supply the fetus while maintaining maternal needs.

Glandular developments

The pituitary gland enlarges during pregnancy. This is presumably a result of its function as the master for all other glandular functions. It must aid in stimulating each of the following:

1. Thyroid function to meet the increased metabolic demands of pregnancy
2. Pancreatic function in the production of increased insulin
3. Ovarian function to aid in hormonal maintenance of the pregnancy
4. Adrenal function to increase cortisol

In addition, the pituitary produces oxytocin and antidiuretic hormone (ADH). Oxytocin improves uterine contractility in labor. ADH aids in increasing fluid volume in pregnancy.

The thyroid gland also enlarges during pregnancy and produces a physiologic goiter. This is a response to the need for increased metabolism. Initially there is increased thyroid binding globulin. Then T_4 uptake increases and T_3 uptake decreases.

The adrenals increase cortisol and catecholamine production. Cortisol contributes to the increase in catecholamines (epinephrine and norepinephrine), which contribute to the increase in maternal cardiac rate. Cortisol also mobilizes glucose and free fatty acids, thereby contributing to improved metabolism.

To counteract some of the effects of vasodilation, the adrenals produce increased amounts of renin and angiotensin. To aid in blood volume expansion aldosterone levels are also increased, contributing to sodium retention.

Renal adaptations

Increased blood volume circulates through the kidneys. This forces glomerular filtration of waste substances to increase. Renal blood flow constitutes about one fifth of the cardiac output. The entire plasma volume is filtered about 60 times per day. Substances to be retained by the body are first filtered and then reabsorbed in the tubules. Substances to be excreted are added to the fluid and flow to the distal portion of the tubule. In pregnancy, larger quan-

tities are filtered in the glomerulus because of the greater capillary pressures associated with the increased blood flow.

In pregnancy, the glomerular filtration rate rises about 50%. Glucose and nitrogenous waste products of metabolism are therefore excreted in the urine in greater quantities. In turn, blood levels of nitrogenous waste products decrease. During pregnancy, normal laboratory values indicating renal function must be adjusted. A blood urea nitrogen (BUN) greater than 13 mg/100 ml is abnormal even though it is within the nonpregnant normal range. Serum creatinine levels greater than 0.8 mg/100 ml also require further investigation of renal function.

Renal function is affected too in pregnancy by anatomic changes. As the gravid uterus displaces other organs in the abdomen, it causes a physiologic hydroureter and hydronephrosis. This is usually more pronounced on the right side than on the left. The dilation of the ureters is further facilitated by estrogen.

During pregnancy the bladder has decreased tone because of hormonal influences. This factor and the distended ureters cause the pregnant woman to be more vulnerable to urinary tract infections. The increased glucose excretion into the urine also promotes bacterial growth.

Hepatic adjustments

In pregnancy there is no change in hepatic size or blood flow. However, the changes in metabolism during pregnancy lead to liver storage and conversion changes. Serum albumin is lower, cholesterol increases 40% to 50%, free fatty acids increase 60%, and phospholipids increase 35% to provide for the nutritional needs of the fetus.

Liver enzymes also reflect changes. While SGOT and SGPT do not change, alkaline phosphatase and leukocyte alkaline phosphatase (LAP) markedly increase.

Hematologic changes

White blood cells, or leukocytes, are primarily responsible for fighting infections. Leukocytes are described either as granulocytes or nongranulocytes. When infection occurs, granulocytes increase and nongranulocytes migrate to inflammatory areas through the circulatory system. During pregnancy the number of neutrophils that are granulocytes increases. This increase is stimulated by estrogen and plasma cortisol.

Normally there is a coexisting potential for coagulation and fibrinolysis. This might also be described as clotting and lysis of clots simultaneously. Coagulation occurs when a platelet comes in contact with a damaged vessel surface.

This contact triggers a cascade of events. First prothrombin activator causes prothrombin to be released in the liver and converted to thrombin. Then thrombin is converted to fibrinogen, which forms fibrin, causing the red blood cells and plasma to mesh. A clot is thus formed. The clot triggers release of activators for plasminogen formation. Plasminogen converts to plasmin and the clot is lysed.

In pregnancy the equilibrium for coagulation-fibrinolysis is skewed toward coagulation. Plasma fibrinogen rises throughout pregnancy. Platelet count can remain normal or can be reduced insignificantly. Circulating activators of plasminogen, however, are reduced, and therefore fibrinolytic activity is diminished. What cannot be explained, however, is the rise in fibrin breakdown products despite apparent diminished fibrinolytic activity in the normal pathways. Factors in pregnancy promoting the increased fibrin breakdown include entry of placental thrombin into maternal circulation, increased amounts of fetoplacental hormones, and perhaps the effect of immunologic complexes present for the fetus.

Gastrointestinal responses

Gastrointestinal changes primarily result from the anatomic shifting of abdominal contents. There is decreased gastric motility and prolonged stomach-emptying time. Constipation is frequently a problem in pregnancy.

The gallbladder is influenced by estrogen and becomes hypotonic. This causes an increased concentration of bile. An increased incidence of [illegible] can result.

Adaptations of the reproductive system

Changes in the reproductive system during pregnancy are the result of increased vascularity and increased hormone production. The vulva and vagina become more vascular during pregnancy. Increased estrogen promotes elasticity. The increased vascularity and elasticity can result in vulvar varicosities. Increased estrogen also increases vaginal secretions and promotes a more alkaline pH, which can predispose to increased vaginal infection.

The cervix becomes softer and shorter and appears cyanotic (Goodell sign). Near term the cervix becomes even softer and shortens because of the influence of prostaglandins released from the stretching uterine musculature.

The muscle of the uterus increases in weight from approximately 60 to 800 g. It does this through hyperplasia, hypertrophy, and stretching. There is a marked increase in uterine vasculature. Vascular resistance is considerably reduced and uterine blood flow increases in the absence of disease. The increased uterine blood flow occurs because of the influences of progesterone, estrogen, and prostaglandins. It is also influenced by vasodilation from the autonomic

nervous system and by the trophoblastic replacement of the muscular and elastic elements in the placental vessel walls.

The ovaries may be enlarged in early pregnancy. The corpus luteum becomes cystic and begins an increased production of progesterone and estrogen to maintain the nutritive lining in the myometrium of the uterus. As the placenta assumes the role of hormone production, by 14 to 16 weeks, the ovaries and corpus luteum return to normal size.

Breast tissue also responds to increased hormonal production of estrogen and progesterone. There is increased vascularity, and veins become more prominent. There is usually an increase in pigmentation and in the size of the areolae. The periareolar glands enlarge to provide greater lubrication during lactation.

DEVELOPMENT OF THE MATERNAL-FETAL UNIT

Knowledge of the growth and development of the maternal-fetal unit provides a basis for the care of the mother and fetus at risk for disease or pregnancy complications. This knowledge can provide a basis for early detection of maternal or fetal problems. Prevention of more serious complications can then ensue. (See also Chapters 9 and 10.)

Embryo

During the luteal phase of the menstrual cycle, cervical mucus becomes receptive to spermatozoa. Ejaculation of sperm into the vagina is aided by mucoid receptivity, which allows rapid migration of spermatozoa through the cervix, into the uterine cavity, and into the fallopian tube.

Active spermatozoa can reach the outer portion of the fallopian tube within 75 minutes. The sperm and ovum meet in the distal portion of the fallopian tube. Fertilization occurs when the sperm penetrates the vitelline membrane of the ovum. Cell division begins, forming a small cell mass called the *morula*.

The morula is passed through the fallopian tube by tubal peristalsis and ciliary propulsion. The outer cell layer of the morula secretes a fluid, which pools in a segmentation cavity. Now the cell mass is called a *blastocyst*.

The blastocyst takes approximately 6 to 7 days to form. Implantation takes place at the blastocyst stage, usually occurring high in the uterine fundus. At this time the outer cells on the blastocyst are called the *trophoblasts*.

The trophoblasts then invade the endometrium. It is thought that the reason that the trophoblast cells are not treated as foreign and rejected by the mother is an exchange of fetal and maternal cytoplasmic and nuclear material from the trophoblastic cells. This allows the maternal immunologic system to tolerate the fetus as a part of the body rather than as foreign to it.

Progesterone from the corpus luteum provides stored nutritive substances

in the endometrium, now called the *decidua*. The trophoblasts secrete proteolytic and cytolytic enzymes, permitting them to destroy vessels, glands, and stroma in the endometrium.

Placenta

The trophoblasts proliferate rapidly after implantation and three layers of cells appear. These send out fingerlike projections called *villi*. The outer layer of cells, or the *syncytiotrophoblast;* the inner layer, or *cytotrophoblast;* and the dividing layer of thin connective tissue, the *mesotrophoblast,* are formed within these fingerlike projections. The mesotrophoblast forms the support for the villi and fetal vascular tissue. The syncytial cells then synthesize proteins, glucose, and hormones for utilization by the embryo.

After 2 or 3 weeks, the chorion begins to develop within the villi. While the chorion is developing, the amnion and its cavity are forming. Two cavities form in the embryonic pole. The ventral cavity is the yolk sac. The dorsal cavity becomes the amniotic cavity. As it enlarges, it forces the formation of the body stalk, the allantois, the blood vessels, and the beginning of the umbilical cord.

The decidua basilis, the layer beneath the embryoblast tissue, comes into contact with the villi, which then multiply rapidly. During villi multiplication, the decidua basilis is called the *chorion frondosum*. By 14 weeks the chorion frondosum organizes into the discrete organ called the *placenta*. The placenta has segments, called *cotyledons,* which are connected by vascular channels to the umbilical cord. The placental surface is exposed to the maternal blood in the intervillous space and thins to a single layer of cells called the *placental membrane*. The exposure of fetal blood to maternal blood across this membrane provides for fetal oxygenation, nutrition, and excretion of fetal wastes. The two umbilical arteries carry carbon dioxide and other wastes from the fetus to the mother. The vein carries nutrition and oxygen to the fetus.

Transfer of oxygen, carbon dioxide, nutrition, and wastes is dependent on molecular size. Smaller molecules such as O_2, CO_2, electrolytes, and water transfer by simple diffusion, moving passively from the side of greater molecular concentration to the side of lesser molecular concentration. Their transfer is largely dependent on the adequacy of uterine blood flow into the intervillous space.

Larger molecules such as glucose are selectively transferred by a more complex process called *facilitated diffusion*. This process occurs against a large concentration gradient and requires a carrier system. Energy expenditure can also provide for selective transfer. Both are more dependent on placental surface area and thickness for their diffusion.

In addition to simple and complex diffusion, the placenta also assumes an